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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/566,856	01/30/2006	Heinz W. Gschwend	17243004001	2175

22511 7590 06/27/2008

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EXAMINER	
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ART UNIT	PAPER NUMBER
1624	

NOTIFICATION DATE	DELIVERY MODE
06/27/2008	ELECTRONIC

Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Notice of the Office communication was sent electronically on above-indicated "Notification Date" to the following e-mail address(es):

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Office Action Summary	Application No.	Applicant(s)	
	10/566,856	GSCHWEND ET AL.	
	Examiner	Art Unit	
	CECILIA M. JAISLE	1624	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE *3* MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *24 March 2008*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-37* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1-10,36 and 37* is/are rejected.

7)☒ Claim(s) *11-35* is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☐ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED OFFICE ACTION

Lack of Unity

Applicants election of Group I without traverse is acknowledged. Claims 1-37 are under examination to the extent they are directed to the Group I elected subject matter.

Rejections under 35 US 112

The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Claims 2-9 and 36 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for inhibition of stearoyl-CoA desaturase activity in mice, does not reasonably provide enablement for treating a disease or condition mediated by SCD in a mammal (claims 2, 3 and 36) or for treating Type II diabetes, impaired glucose tolerance, insulin resistance, obesity, fatty liver, non-alcoholic steatohepatitis, dyslipidemia or metabolic syndrome (claims 4-9). The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to use the invention commensurate in scope with these claims.

Many factors require consideration when determining whether sufficient evidence supports a conclusion that a disclosure satisfies the enablement requirement and whether any necessary experimentation is “undue.” MPEP 2164.01(a). These factors

include: (1) the claim breadth; (2) the nature of the invention; (3) the state of the prior art; (4) the level of predictability in the art; (5) the amount of direction provided by the inventor; (6) the presence of working examples; and (7) the quantity of experimentation needed to make or use the invention based on the content of the disclosure. *In re Wands*, 8 USPQ2d 1400, 1404 (Fed. Cir. 1988)(reversing the PTO's determination that claims directed to methods for detection of hepatitis B surface antigens did not satisfy the enablement requirement). See also *In re Goodman* 29 USPQ2d 2010, 2013 (Fed. Cir. 1993). Application of these factors to the present application supports the determination that the present disclosure fails to satisfy the enablement requirement:

(1) Breadth of claims.

(a) Scope of the compounds. The claims cover potentially millions of pyridazines compounds of Formula (I).

(b) Scope of the diseases covered. The claims embrace treating all diseases, including ones as yet undetermined, which are mediated by SCD in a mammal (claims 2, 3 and 36), including Type II diabetes, impaired glucose tolerance, insulin resistance, obesity, fatty liver, non-alcoholic steatohepatitis, dyslipidemia or metabolic syndrome (claims 4-9). Therefore, the claims are of unknown and indeterminate scope.

Current medical knowledge emphasizes that each specifically named disease/condition requires lifestyle changes, especially diet and exercise, for successful treatment. The major goal in treating Type II diabetes is to minimize blood sugar elevation without causing abnormally low blood sugar levels. Type II

diabetes is treated first with weight reduction, diabetic diet, and exercise. When these measures fail to control the elevated blood sugars, oral medications are used. If oral medications are still insufficient, treatment with insulin is considered.

Impaired Glucose Tolerance (IGT) is a pre-diabetic state dysglycemia, that is associated with insulin resistance and increased risk of cardiovascular pathology. Although some drugs can delay the onset of diabetes, lifestyle modifications play a greater role in the prevention of the disease. Patients identified as having an IGT should exercise regularly and have a balanced diet removing sugar.

Reducing insulin need and increasing cell sensitivity to the action of insulin is generally the management for insulin resistance. Recommended treatment involves dietary therapy, which should be low- or very-low calorie and low-fat, and physical activity, to maintain weight loss and reduce waist circumference.

Treatment of fatty liver disease depends on the underlying cause. If the underlying cause is high level of alcohol consumption, treatment includes limitation or elimination of alcohol consumption. If alcohol consumption is not the cause (non-alcoholic steatohepatitis or NASH), preferred treatments include weight loss and exercise, diabetes control, cholesterol control and avoidance of toxic substances, including alcohol and avoidance of medications and other substances that can cause liver damage.

Dyslipidemia requires lipid lowering with aggressive statin therapy, and combined therapy including bile acid resins, nictonic acid, fibrates and fish oil or omega-3 fatty acids.

An association between certain metabolic disorders and cardiovascular disease is known as metabolic syndrome; a clustering of risks leading to cardiovascular disease include insulin resistance, hypertension, cholesterol abnormalities, and an increased clotting risk. Patients are most often overweight. A majority of people with metabolic syndrome are overweight and lead a sedentary lifestyle. Lifestyle modification is the preferred treatment. Weight reduction usually requires a specifically tailored multifaceted program that includes diet and exercise. Sometimes medications or surgery may be useful.

(2) The nature of the invention and predictability in the art: The nature of the invention is therapeutic use of the inventive compounds to treat a plethora of diseases/conditions set forth in the claims.

Dobrzyn, recognizes:

> Taken together, the findings reveal SCD to be a promising therapeutic target for the treatment of obesity, diabetes, liver steatosis and other metabolic diseases. However, the potential use of an SCD inhibitor as a human therapeutic agent awaits a more compete understanding of the mechanism underlying the effects of SCD deficiency and indication that the inhibition of this enzyme is both safe and efficacious.

It is well established that "the scope of enablement varies inversely with the degree of unpredictability of the factors involved;" physiological activity is generally considered to be an unpredictable factor. *In re Fisher*, 166 USPQ 18, 24 (CCPA 1970).

(3) Direction or Guidance: The direction and guidance provided is very limited. The dosage range information (pages 31-34, *inter alia*) is so meager, that it would require extensive experimentation to determine a specific dosage for a specific disease/condition, mode of administration and therapeutic regimen. Moreover, the

dosage is generic; the same for the many disorders covered by the specification. Thus, there is no specific direction or guidance regarding a regimen or dosage effective specifically for the various types of diseases/conditions claimed. *In vivo* testing is limited to mice, so claim 3, directed specifically to human use, has no guidance in the specification or correlative teachings in the art which can be extrapolated for support of treatment of humans.

(4) State of the Prior Art: Applicants do not provide highly predictive competent evidence or recognized tests to treat all conditions and other diseases recited for theinventive compounds. Pharmacological activity in general is unpredictable. In applications involving physiological activity, such as the present,

> "The first paragraph of 35 U.S.C. 112 effectively requires that the scope of the claims must bear a reasonable correlation to the scope of enablement provided by the specification to persons of ordinary skill in the art."

Plant Genetic Systems N.V. v. DeKalb Genetics Corp., 65 USPQ2d 1452, 1456 (Fed.Cir. 2003).

At present no known drug can successfully treat all of the conditions/diseases recited and encompassed by the present claims, despite the fact that many drugs are said to inhibit stearoyl-CoA desaturase activity. Substantiation of utility and its scope is required when utility is "speculative," "sufficiently unusual" or not provided. See *Ex parte Jovanovics, et al.*, 211 USPQ 907, 909 (BPAI 1981). Also, note *Hoffman v. Klaus*, 9 USPQ2d 1657 (BPAI 1988) and *Ex parte Powers*, 220 USPQ 924 (BPAI 1982) regarding types of testing needed to support *in vivo* uses.

See the discussion of Dobrzyn above. In addition, Giutiérrez-Juárez reports:

> In the effects of SCD1 deficiency are confirmed in humans, the pharmacological inhibition of this enzyme should have independent and beneficial effects on both weight gain and insulin action. SCD1 deficiency may also account for the lack of hepatic insulin resistance in several genetic models in which a primary alteration in other steps in hepatic lipid metabolism also leads to a secondary decrease in SCD1 expression.

Thus, the state of the prior art recognizes that the inhibition of stearoyl-CoA desaturase activity as an effective treatment of the specific diseases and conditions recited is an area for future research, especially as applied to human therapy.

(5) Working Examples: The specification provides enablement for inhibition of stearoyl-COA desaturase activity in mice (pages 31-35 and 50-52, *inter alia*) and claims directed thereto would be deemed allowable.

Although the specification refers to testing procedures, it is apparent that the only testing actually performed is with mice, because that is the only specific data reported. In addition, the specification tests offer no evidence establishing any connection between inhibition of stearoyl-COA desaturase activity in mice and any specific disease or condition recited in the claims, especially as applied to human patients. The stearoyl-COA desaturase activity testing in mice (pages 50-52, *inter alia*) provides no correlation between any specific compound of the present invention and inhibition of any specific disease or condition. There is no indication that the testing reported in the present specification is art-recognized.

(6) Skill of those in the art: The prior art recognizes that no compound has ever been capable of treating all diseases or conditions recited by the present claims generally.

Discussions above of the skill of those in the art support that successful treatment of diseases caused by and/or associated with stearoyl-CoA desaturase activity is a subject for further investigation, especially as applied to human patients.

(7) The quantity of experimentation needed: Based on the content of the disclosure, an undue burden would be placed on one skilled in the pharmaceutical arts to use the invention, since the disclosure gives the skilled artisan inadequate guidance regarding pharmaceutical use, for the reasons stated above.

Consideration of the above factors demonstrates that the present application sufficiently lacks enablement of the present claims. In view of the breath of the claims, the pharmaceutical nature of the invention, unpredictability of relationship between stearoyl-CoA desaturase activity and specific conditions and diseases, one of ordinary skill in this art would have to undergo an undue amount of experimentation to use the instantly claimed invention commensurate in scope with the claims.

MPEP 2164.01(a) states,

> A conclusion of lack of enablement means that, based on the evidence regarding each of the above [Wand] factors, the specification, at the time the application was filed, would not have taught one skilled in the art how to make and/or use the full scope of the claimed invention without undue experimentation. *In re Wright*, 27 USPQ2d 1510, 1513 (Fed.Cir. 1993).

The above consideration justifies that conclusion here; undue experimentation would be required to practice Applicants' invention.

Reply to Mar. 24, 2008 Remarks

Ntambi, cited by Applicants, emphasizes the need for further research, "These metabolic changes recommend SCD as a promising therapeutic target for the many disorders associated with the metabolic syndrome." When a drug merely has promise, it is not yet enabled.

Park, cited by Applicants, noted several concerns regarding obesity treatment by SCD-1 inhibition:

- "The variation in concentration in serum lipids for the same strains reported by different laboratories may be related to differences in diet compositions because fatty acids and other ingredients vary among preparations of unpurified diets."
- "Others found strain … -specific differences in serum apolipoprotein E concentrations after overnight food deprivation in mice fed unpurified vs. a highly atherogenic diet, suggested our results may be confounded because we analyzed expression 2 h postprandially."
- "Diet composition or time between the last meal and time of killing may have been responsible for the observed differences between our studies and others."
- "Although significant correlations show associations, it is not clear whether these interactions were directly linked or whether they correlated because of independent linkages with other common factors."
- "A small but growing number of disease QTL have been identified in humans and in laboratory animals …, but many possible loci remain unidentified because of the number of possible nutritional and genetic combinations yet to be analyzed."

Park concludes with a recommendation for further research: "Additional experiments are required to test whether Scd1 or other diet-regulated genes are involved in the molecular mechanism of disease."

Miyazaki I, et al., J. Lipid Res., cited by Applicants, acknowledges varying effects of different SCD genes: (1) "[D]espite the induction of SREBP-1 by a lipogenic diet and the expression of SCD2 in liver of the SCD-/- mice, the levels of triglycerides remained low, suggesting that SCD2 could not compensate for the SCD1 deficiency." (2) "[T]he present work demonstrates that dietary induction of triglyceride synthesis in mouse liver is highly dependent on the expression of the SCD1 gene. SCD2 cannot compensate for the deficiency." The presently claimed methods recite treatment of disease by mediation of SCD without regard to the particular isoform. Miyazaki I finally concludes: "[T]he regulation of SCD may have broad implications for its potential use as a target in the treatment of human hypertriglyceridemia." The language “may have broad implications for its potential use” clearly indicates that the material is not yet enabled.

Miyazaki II, et al., J. Nutrition, cited by Applicants, notes: "[T]he alteration of SCD activity in the eyelid can be implicated in human eye diseases." Miyazaki II likewise conclude with the requirement for further research; "The studies described here may have broad implications for potential use of the SCD1 gene as a target in the treatment of human eye and skin diseases." At that, Miyazaki II is limited to only very certain types of eye and skin diseases with very specific causes.

Attie, cited by Applicants, notes factors other than stearoyl CoA desaturase activity are triglyceride production rate-limitors: "The finding that the mean 18:1/18:0

ratio increased in the group with a triglyceride reduction on the high carbohydrate diet suggests that other metabolic response may have acted to reduce triglyceride production and/or increase triglyceride clearance in these individuals." Attie notes the need for more research: "This, together with the lipid profiles of the SCD-deficient mice, suggests that SCD might be an attractive target for triglyceride-lowering drugs." Attie clarifies that enablement is just a possibility, not yet achieved.

Regarding acne treatment by SCD inhibition, Zheng, cited by Applicants, observe: "Although the exact mechanism is unclear at present, fatty acids are known mediators of signal transduction and have been implicated in acne." Zheng does not state that stearoyl-CoA desaturase-1 suppression would be expected to help with acne, and “fatty acids” is a very broad term. Zheng observes a distinction between mice and human subjects: "We are unaware of a direct parallel between the ab mutation [in mice] and a human disorder, but the small sebaceous glands and associated scarring alopecia of mutant mice are reminiscent of some of the clinical scarring alopecias."

Sjogren, cited by Applicants, observes: "Our results suggest that elevated SCD activity in adipose tissue could contribute to the development of insulin resistance. … [I]t is also possible that increases in adipose tissue SCD activity are merely a consequence of the development of insulin resistance." This indicates that even as of 2008, the relationship cannot be firmly stated, as the author uses hedging terms: “suggest”, ”could contribute” and “is also possible”.

Warensjo, cited by Applicants, reveals, "The number of observations was much lower (n=611) for the estimated SCD activity than for other variables since the fatty acid

analyses were only carried out in a randomly selected subsample." In addition, the Warensjo studies contain the following cautions:

> First, fatty acid composition was only assessed in a subsample of the study population suggesting decreased power. Second, SCD activity is estimated and is not a true enzyme activity. Third, the activity was estimated in serum cholesterol esters and not in target tissues, i.e., adipose tissue, liver or skeletal muscle.

Warensjo acknowledged limitations of their study, "First, the cohort consisted of only men and the data are observational. Second, even though the 8 SNPs examined in this study adequately capture most of the genetic variability in the SCD1 gene, it should be noted that we might lack possible genetic variation."

Thus, the state of the prior art recognizes that the inhibition of stearoyl-CoA desaturase activity as an effective treatment of the specific diseases and conditions recited is an area for future research, especially as applied to human therapy.

Rejections Under 35 USC 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

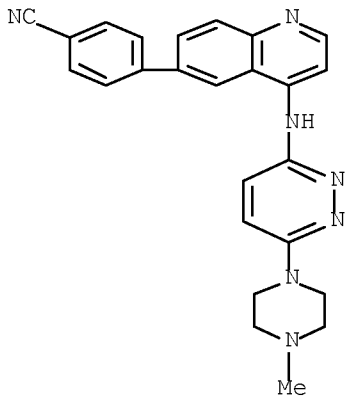
This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein

were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(e), (f) or (g) prior art under 35 U.S.C. 103(a).

The factual inquiries set forth in *Graham* **v.** *John Deere Co.*, 383 U.S. 1, 148 USPQ 459 (1966), that are applied for establishing a background for determining obviousness under 35 U.S.C. 103(a) are summarized as follows:

1. Determining the scope and contents of the prior art.
2. Ascertaining the differences between the prior art and the claims at issue.
3. Resolving the level of ordinary skill in the pertinent art.
4. Considering objective evidence present in the application indicating obviousness or nonobviousness.

Claims 1, 10 and 37 are rejected under 35 U.S.C. 103(a) as being unpatentable over Dickson, et al., WO/2005/120509, entitled to the Jun. 4, 2004 priority date, describing RN 871873-26-6, Benzonitrile, 4-[4-[[6-(4-methyl-1-piperazinyl)-3-pyridazinyl]amino]-6-quinolinyl]-,



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exhibiting ATP-utilizing enzyme inhibitory activity and useful for treatment of Alzheimer's

disease, stroke, diabetes, obesity, inflammation, Crohn's disease, cancer, etc. Although this compound is excluded by the amendment limiting R11 to C1-C3alkyl, the compound of Dickson renders obvious lower alkyl homologs thereof, encompassed by the present claims when R11 is hydrogen. The skilled chemist would be well motivated to prepare compounds and their compositions homologous with that of Dickson according to the procedures taught therein with the expectation that such compounds would have the same activity taught by Dickson.

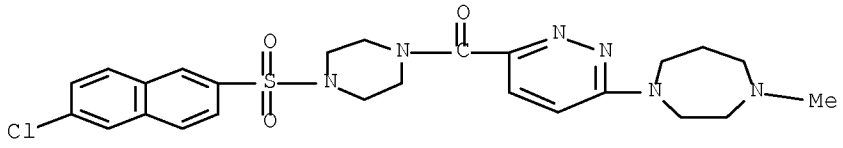
It would have been obvious to one of ordinary skill in the art at the time the present invention was made to modify the compound of Dickson to prepare compounds homologous therewith. One of ordinary skill in the art would have been motivated to prepare the instantly claimed compounds because such structurally homologous compounds are expected to possess similar properties to the Dickson compound. It has been held that compounds that are structurally homologous to prior art compounds are *prima facie* obvious, absent a showing of unexpected results.

> An obviousness rejection based on similarity in chemical structure and function entails the motivation of one skilled in the art to make a claimed compound, in the expectation that compounds similar in structure will have similar properties.

In re Payne, 203 USPQ 245, 254 (CCPA 1979). See *In re Papesch*, 137 USPQ 43 (CCPA 1963) and *In re Dillon*, 16 USPQ2d 1897 (Fed.Cir. 1991) (discussed in MPEP § 2144) for an extensive case law review of obviousness based on close structural chemical compound similarity. See MPEP § 2144.08, ¶ II.A.4(c). Compounds which are homologs (compounds differing regularly by the successive addition or subtraction of the same chemical group, e.g., by -CH3 or lower alkyl groups), as here, are generally

of sufficiently close structural similarity that there is a presumed expectation that such compounds possess similar properties. *In re Wilder*, 195 USPQ 426 (CCPA 1977).

Claims 1, 10 and 37 are again rejected under 35 U.S.C. 103(a) as being unpatentable over Herron, et al., WO 2002010154, published Feb. 7, 2002, describing RN 395684-35-2, Piperazine, 1-[(6-chloro-2-naphthalenyl)sulfonyl]-4-[[6-(hexahydro-4-methyl-1H-1,4-diazepin-1-yl)-3-pyridazinyl]carbonyl]-,



, useful in the treatment of thromboembolic disorders. Although this compound is excluded by the amendment limiting x and y to 1 and limiting R11 to C1-C3alkyl, the compound of Herron renders obvious lower alkyl homologs thereof, encompassed by the present claims when x is 2 and R11 is hydrogen. The skilled chemist would be well motivated to prepare compounds and their compositions homologous with that of Herron according to the procedures taught therein with the expectation that such compounds would have the same activity taught by Herron. The discussion above of the obviousness of homologous compounds is repeated here as equally appropriate.

Objected Claims – Allowable Subject Matter

Claims 11-35 are objected to as being dependent upon a rejected base claim, but would be allowable if rewritten in independent form including all of the limitations of

the base claim and any intervening claims. An examiner's statement of reasons for indication of allowable subject matter can be found in the Office Action of Sep. 24, 2007.

Conclusion

Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL**. See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Cecilia M. Jaisle, J.D. whose telephone number is 571-272-9931. The examiner can normally be reached on Monday through Friday; 8:30 am through 5:00 pm.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, James O. Wilson can be reached on 571-272-0661. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

/James O. Wilson/
Supervisory Patent Examiner, Art Unit 1624

Cecilia M. Jaisle, J.D.
6/10/2008